Student Clinical Paper

Hydropic Conditions of the Bovine Uterus

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Hydropic conditions of the bovine uterus are encountered sporadically by the bovine practitioner. Although hydrops is not an everyday occurrence, it is important to recognize the condition so the appropriate medical treatment can be instituted. Accurate diagnosis and prompt appropriate treatment can allow many cows to be salvaged. The most common clinical sign observed with uterine hydrops is abdominal distension. This condition is frequently misdiagnosed as indigestion, bloat, traumatic gastritis or multiple fetuses.[1] The sudden increase in weight and volume predisposes affected cows to ventral abdominal muscle herniation, prepubic tendon rupture or coxofemoral dislocation. The fluid can prevent the animal from lying in a normal position and cause backward extension of the rear limbs when the animal is in sternal recumbency. It may interfere with the animal's ability to rise.[2] Dystocia may result due to uterine inertia and the fetus is usually dead at birth or is born weak and dies shortly thereafter. Retention of fetal membranes and septic metritis are common sequelae.[1] The prognosis for the life of the cow, especially with secondary complications, is poor.

Hydrallantois and hydramnion are the two forms of uterine hydrops. Hydrallantois accounts for 90% of uterine hydrops while hydramnios accounts for 10%.[3] In addition to the difference in incidence there are great differences in the mechanisms, clinical signs, diagnostic signs, sequelae and prognosis between these two conditions.

Hydrallantois usually affects cows at 3 or more years of age. Hydrallantois is often associated with a diseased uterus in which most of the caruncles in one horn are not functional and the remainder of the placentomes are often greatly enlarged and possibly diseased. Portions of the placenta may be necrotic and edematous. There is an apparent structural or functional change in the allantois chorion and its vasculature which lead to a collection of a transudate that more closely resembles plasma than normal allantoic fluid. Some have felt that an abnormality of the fetal kidneys may also contribute to this condition; however, one author feels that any changes found in the fetus are secondary to the primary placental dysfunction.[1] Although definitive causes of hydrallantois have not been identified, an association may also exist between hydrallantois and consumption of damaged forages. A possible lack of vitamin A in the forages could result in a lowered resistance of the endometrium to disease. Although rarely in heifers, hydrallantois may result from a congenital lack of uterine caruncles.[1]

General signs seen with hydrallantois are anorexia, lack of rumination, excessive water intake and restlessness. The characteristic signs of hydrallantois include rapid abdominal enlargement (over 5 to 20 days), occurring as early as the fifth month of gestation in severe cases. Spontaneous abortion at 6 to 9 months may frequently be observed. In severe cases the fluid may reach 30 to 60 gallons with the abdomen becoming distended, tense and barrel-shaped.[1] On rectal palpation the uterus is greatly distended and fills the entire abdominal cavity. The placentomes and fetus are difficult to palpate and are identified through the tense uterine wall. In mild cases the condition may not be diagnosed until parturition when an excessive amount of clear, watery, amber transudate is expelled.

Hydramnios is associated with a genetic or congenitally defective fetus. The accumulation of fluid is in part associated with failure of the fetus to swallow amniotic fluid.[1] Among the conditions resulting in hydramnois are "bull dog" fetuses in Dexter cattle, muscle contracture monsters in Red-Danish cattle, and hydrocephalic calves in Herefords. Hydramnios is often seen in hybrids, such as crosses between American bison and domestic cows.[2]

Hydramnios develops gradually over several months during the latter half of pregnancy. There is an accumulation of approximately 50 to 30 gallons of amniotic fluid. The slow, gradual onset allows the uterus to accommodate the distension and the placentomes and the fetus are usually palpable per rectum. Many cases are diagnosed or only suspected at time of calving due to a large amount of syrupy, viscid fluid that often contains meconium.[1] Since the abdominal wall has more time to adjust to the increased weight and volume, the abdomen is pear-shaped and less tense than that of a cow with hydrallantois. Dystocia at parturition is not uncommon due to the enlarged uterus, uterine inertia and defective fetus. Abortions and premature parturitions are fre-

quent. Retention of the placenta and metritis are not serious problems as with hydrallantois. The fetus is invariably defective and dies. The prognosis for future breeding of the dam is fair to good.

Alterations in electrolyte and hormone concentrations in maternal plasma and fetal fluids also occur in hydrops. The ionic composition of allantoic fluid loses its initial resemblance to extracellular fluid between the 2nd and 4th months of normal pregnancy.[4] In hydrallantois, the excretion or reabsorption mechanisms appear to be altered or ineffective. Average potassium and creatinine levels were elevated in samples of amniotic fluid obtained from 3 cows at calving. Sodium and chloride in the allantoic fluid were also elevated in the 3 cow study while creatinine levels were lower than normal.[4] A structural or functional change in the chorioallantoic membrane could be the cause with some involvement of the fetal renal tubules secondarily. Low peripheral plasma concentrations of estradiol, normally produced by the placenta in late pregnancy, were detected in the same study. The estradiol concentrations may have been associated with the altered ionic composition of the fetal fluids. It has been hypothesized that in the late gestation cow estradiol synthesis is directly responsible for maintaining membrane permeability to ions. Defects in chorio-allantoic function or hormonal-controlled permeability to ions may occur concurrently.[4] It has also been suggested that abnormal secretion of cortisol from the fetal adrenal gland may be responsible for the changes in membrane permeability. More work is needed to examine fetal membrane structure and function, especially with respect to steroid synthesis and membrane permeability. In addition, further examination of fetal pituitary - adrenal activity would be beneficial.[4]

Treatment of cases of hydrops varies with the severity and duration of the condition. In severe cases with a closed cervix, the prompt termination of the pregnancy is desirable. Slaughtering the affected animal before parturition should be considered. Since the cause of hydrallantois is uterine or placental dysfunction, recurrence in the cow in future pregnancies is a definite possibility. The genetic implications should be considered before treating cows with hydramnios.

The best treatment in cows that are not slaughtered, regardless of cause, is to induce abortion or calving. This may be done by using glucocorticoids such as dexamethasone at a dosage of 20-40 mg IM or flumethasone at a dosage of 10-20 mg IM; or through the administration of prostaglandin $F_2\alpha$ at a dosage of 25-35 mg IM. Concomitant administration of glucocorticoids and prostaglandins will provide the most effective induction regimen.[2]

It is important to closely monitor the cow and examine her by vaginal palpation at 12 hour intervals. When the cervix has dilated to the point that a hand can be passed through the dilated cervical canal, the fetal membranes should be ruptured and the fluid released. Trocharization of the distended uterus through the abdominal wall to slowly drain uterine fluids and relieve uterine distension, is not recommended. It is not unusual for parturition to cease after the calf has entered the vaginal canal. Vaginal exams should be continued until the calf is delivered by assisted extraction, fetotomy, or cesarean section.[3] Elective cesarean section has been attempted but has met with limited success and often holds a poor prognosis because the uterus is atonic, thin walled and friable.

There is some disagreement over the need for rapid infusion of large volumes of intravenous fluids at the time of delivery. Some authors believe that the fluids can aid in the prevention of shock that may occur with the rapid release of placental fluids.[1] Others believe that this fluid loss is from the uterus and not the circulatory system and therefore they are not in need of fluid support.[2] Fluid therapy is indicated to correct electrolyte and fluid imbalances in the cow that result either directly from the hydrops condition and sequestration of fluids in the uterus or from gastrointestinal dysfunction or inappetence caused by the hydrops. This therapy should be initiated immediately and monitored throughout the course of induction and parturition / abortion. Aftercare and followup support are necessary in cases of surgical cesarean section intervention. Therapy is often necessary for the anticipated sequelae of retained placentas and metritis. Counseling on future reproductive use and genetic selection of the animal is recommended.

References

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